IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

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| Applicant | : Cheng Hwang et al. | Art Unit | : 1617 |
| Serial No. | : 10/721,118 | Office | : Gina C Yu |
| Filed | : November 25, 2003 | Conf. No. | : 6141 |
| Title | : REDUCTION OF HAIR GROWTH | | |

**Mail Stop Appeal Brief - Patents**
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

BRIEF ON APPEAL

**(1) Real Party in Interest**

The real party in interest is The Gillette Company, Prudential Tower Building, Boston, Massachusetts. The Gillette Company is owned by The Procter & Gamble Company.

**(2) Related Appeals and Interferences**

There are no related appeals or interferences.

**(3) Status of Claims**

All pending claims (claims 1, 2, 4, 8 and 29-25) were finally rejected in an office action mailed September 16 , 2008 and no claim amendments have been made after that final rejection. Remarks but no claim amendments were filed on November 3, 2008. An Advisory Action was mailed December 17, 2008.

Claim 3 is cancelled.

Claims 5-7, 9-28 and 46 are withdrawn.

**(4) Status of Amendments**

No claim amendments are pending.

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**(5) Summary of Claimed Subject Matter**

Applicants have discovered that agonists of a cell surface feature known as the prostaglandin DP-receptor (specification at page 3, line 27) can be applied to an area of mammalian skin to reduce hair growth (specification at page 2, line 2 under "Summary"). Claim 1 reads as follows:

1. A method of reducing mammalian hair growth which comprises

selecting an area of skin from which reduced hair growth is desired; and

applying to said area of skin a dermatologically acceptable composition comprising an agonist of prostaglandin DP-receptor in an amount effective to reduce hair growth.

**(6) Grounds of Rejection to be Reviewed on Appeal**

All pending claims are rejected as obvious from a combination of two references: Billoni et al. Aeta Derm. Venereol. 2000 80:329-334 ("Billoni") in view of Monneret et al. J. Immunol. 2002 168:3563-3569 ("Monneret").

**(7) Argument**

As noted, claim 1 features methods of reducing hair growth by topically applying an agonist of the DP-receptor. To understand this appeal, it is important to:

a) know some basic facts about the DP-receptor; and

b) understand that there is no relationship between the DP receptor and an entirely different class of receptors known as PPAR receptors.

We begin with a short Background.

**I. Background**

Prostaglandins are very powerful molecules which are sometimes classified as a type of hormone and which have far reaching and varied effects on the body. Among other things, prostaglandins work by interacting with structures (receptors) on the surface of various cells, causing those cells to undergo certain changes. In other words, prostaglandins are "ligands" for certain cell-surface receptors known as PG or prostanoid receptors.

The specification includes a table at page 2 listing four specific prostaglandins, matching each with its receptor.

| Prostaglandin (PG) or Prostanoid | G-protein-coupled PG or Prostanoid receptor* |
|---|---|
| $PGE_2$ | EP (EP1, EP2, EP3 and EP4) |
| $PGF_{2\alpha}$ | FP |
| $PGI_2$ | IP |
| $PGD_2$ | DP |

*The receptors are distinguished by their ligand-binding profiles, and the signal transduction pathways activated on ligand binding.

## II. The Invention

The invention specifically features the use of agonists of the DP receptor, the fourth receptor listed in the above table. The natural ligand for the DP receptor is a molecule known as $PGD_2$.

It is critical to understand that the Office cites prior art which focuses on an entirely different receptor family, the PPAR receptor family, which includes three different receptors designated $\alpha$, $\delta$ and $\gamma$. These two cell structures (the DP receptor structure called out in the claims and the PPAR receptor family that concerns the art) are different. For our purposes, it is enough to know that the DP receptor of the claims occurs on the **outer cell membrane** and it respond to $PGD_2$, initiating a number of intra-cellular events. In contrast, members of the PPAR receptor family studied in the prior art occur on the cell's **nuclear membrane**. Neither the Office nor the cited art contends that these structures are the same.

With that background, we look at the prior art underlying the obviousness rejection.

## III. The Prior Art

### A. The only findings in the art about hair growth concern PPAR receptors, not the DP receptor, and those findings are based on contradictory findings about a single PPARα agonist, clofibrate.

Below we review each sentence of the final rejection regarding hair growth.

*Billoni teaches expression of [PPARs] in human hair follicles*

This statement is true but irrelevant. PPARs are entirely different structures from DP receptors.

> *[Billoni] indicates that high clofibrate concentration...led to cessation of hair follicle growth...[and] that an unbalanced lipid metabolism can lead to an alteration of the human hair cycle, and suggests that this alteration is at least partially due to altered PPAR-controlled pathways. [Final Rejection p. 2, lines 14-18]*

Here the Office points to the only experimental evidence in the prior art about hair growth [Billoni p.333], which concerns a single PPARα agonist known as clofibrate. This finding does relate to hair growth, but the teaching is entirely limited to PPAR-controlled events. All the art says is that clofibrate is a PPARα agonist. The Office makes no finding that it is a DP receptor agonist.

Even if Billoni's report about the effects of clofibrate had some bearing on the claimed invention, the Office ignores important teachings about whether in fact clofibrate causes cessation of hair growth or, to the contrary, clofibrate enhances hair growth. Here are quotes from Billoni the Office ignores [Billoni, left col. lines 21 et seq.]:

> [Two] effects [of clofibrate] were observed [in cultured hair follicles]: while high clofibrate concentration ($10^{-6}$ M) led to cessation of hair follicle growth, low clofibrate concentrations ($10^{-6}$ M – $10^{-8}$ M) **enhanced the *in vitro* survival of human hair follicles**. This effect on survival suggests that **clofibrate may have a beneficial effect on hair growth**, albeit with in a narrow concentration window. **The cause of hair growth cessation observed with $10^{-6}$ M clofibrate remains** obscure but probably cannot be accounted for by a toxic effect...[Emphasis is supplied].

So when the Office finally gets around to making a finding about the effect of clofibrate on hair growth, the finding ignores what the Billoni authors actually said. Note in particular the emphasized sentences in the above quotations. When it comes to clofibrate, the authors are clear that the clofibrate has contradictory effects on hair growth. They hypothesize that the contradiction might be explained by differences in concentration but conclude that the reasons for their observation **remain obscure**.

The Office picks and chooses parts of Billoni and makes no attempt to draw an impartial teachings from the reference as a whole. Specifically, the Office ignores the finding that the molecule in question, clofibrate, can enhance hair growth.

Undeterred by the equivocal basis for the finding on the effects of clofibrate, the Office then extrapolates far beyond clofibrate to other molecules with structures that are unrelated to clofibrate and that operate on other receptors.

**B. The art has no finding that a PPAR-γ agonist, 15d-J2, inhibits hair growth**

The Office builds on the unsound conclusion about clofibrate and implies, but does not say, that a second molecule, 15d-J2, also inhibits hair growth. Billoni reports no experiments about hair growth using 15d-J2 or any molecule other than clofibrate, a fact which the Office does not challenge. In fact, Billoni says nothing about whether 15d-J2 inhibits hair growth.

On the subject of 15d-J2, the Office nevertheless concludes that "[Billoni] teaches that [15d-J2] is a potent adipogenesis inducer and PPAR-γ activator." This statement is irrelevant to hair growth, and adipogenesis induction is related to PPARs and not to DP receptors.[1]

In a key part of the analysis underlying the rejection, the Office says [Final Rejection, page 2, lines 19-21]:

> *Billoni suggests that ligands other than clofibrate, that are specific for PPAR-δ and -γ expressed in human hair follicles would confirm similar results. See Discussion.*

Once again, it is important to see what the cited portion of Billoni actually says (page 333, left col. lines 47 et seq.):

> Although these effects on human hair [reports that certain substances alter hair growth] have been known for a long time, the **putative direct effect of these agents on the hair cycle remains to be demonstrated**. Taken together these clinical studies clearly demonstrate that an unbalanced lipid metabolism can lead to an alteration of the human hair cycle. **We can hypothesize** from our results that the alteration in the hair cycle **might be at least partially due to altered PPAR-controlled pathways**. This could be confirmed by using ligands other than clofibrate which are specific for the 2 other PPARs (-δ and -γ) expressed in the human hair follicles. [Emphasis is supplied.]

[1]One PPAR function is creating more peroxisomes, intracellular organelles that metabolize fatty acids and many other metabolites. This PPAR function gave PPARs their name, Peroxisome Proliferation-Activated Receptors. Peroxisomes are bounded by a single membrane that separates their contents from the cytosol (the internal fluid of the cell). They contain membrane proteins critical for various functions, such as importing proteins into the organelles and aiding in proliferation.

Comparing the Office finding with the above quotation, it is clear that the Office mischaracterizes Billoni. Billoni invites experiments to confirm its hypothesis about PPAR's involvement in the obscure events underlying the clofibrate experiments. The Office concludes that this single sentence inviting basic experiments on mechanism is "sufficient motivation to make the use of a PPAR-γ activator in regulating hair growth..." [Final Rejection page 3, lines 18-20]. So the Office doesn't really have any finding, or basis for a finding, about whether other PPAR agonists in general (or PPAR-γ agonists or 15d-J2 in particular) might inhibit hair growth.[2] The above-quoted statement in Billoni is no more than speculation about future experiments that might be conducted.

We note in passing that the Office appears to ignore the difference between PPARα and PPARγ. These two structures are members of the same family but they have important structural and functional differences that the Office ignores. The only equivocal evidence in the cited art about hair growth control concerns a PPARα agonist. There is no evidence one way or another for a PPARγ agonist reducing hair growth.

In sum, 15d-J2 is a PPARγ agonist, but no cited art "suggests", much less says, that 15d-J2 affects hair growth by interacting with a member of the PPAR receptor family or by interacting with the DP receptor or by any other mechanism.

### C. The art has no finding that $PGD_2$ inhibits hair growth

From here on, the basis for the rejection moves from speculation into the world of pure conjecture. The Office concludes that Monneret teaches that $PGD_2$ is a precursor to 15d-J2. That may be, but as we have seen, there is no basis for the finding that 15d-J2 causes cessation of hair growth.

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[2] The Office further characterizes Billoni as teaching that a high concentration of PPARα leads to cessation of hair growth. [Final Rejection page 3, lines 5-6]. Billoni has no such teaching. Billoni does teach that PPAR receptors are expressed in hair follicles. The Office does not cite a statement from Billoni about the high concentration of such receptors. The Office apparently meant to reiterate his earlier finding that a high concentration of one particular PPARα **agonist,** clofibrate, caused a cessation of hair growth. As detailed above, in drawing conclusions from that teaching, the Office ignores important portions of the Billoni article.

While the Office doesn't say so, it appears that the logic of the rejection might be that $PGD_2$ inhibits hair growth. There is no mention in either reference of the DP2 receptor in connection with hair growth and no mention that $PDG_2$ inhibits hair growth.

**D. The art has no finding that 15d-$PGD_2$ inhibits hair growth**

The fourth molecule at issue is as 15-deoxy-$\Delta^{12, 14}$ $PGD_2$ ("15d-$PGD_2$"), which is an agonist of the DP2 (see claim 8).

The problem is, neither Billoni nor Monneret says anything at all about whether 15d-$PDG_2$ has an effect on hair growth. The rejection is based on the chain of inferences outlined above, not on any evidence or statements in the art.

As noted, the original link in the chain relating to clofibrate is flawed. From there, the rejection proceeds with layer-upon-layer of unsupported inferences. Here is how the Office expresses the build up to the key finding (quoting the Final Rejection page 3, lines 4-8):

> *1) Billoni teaches that a high concentration of PPAR-alpha [sic a high concentration of the PPAR-alpha agonist clofibrate?] leads to cessation of hair follicle growth in vitro, and suggest [sic suggests] that ligands specific for PPAR-$\delta$ and -$\gamma$ would yield similar results;*
>
> *2) the reference [Billoni] also teaches that 15d-J2 is a PPAR-$\gamma$ activator; and*
>
> *3) Monneret teaches that PDG2 is an analog of PGD2, which is a precursor to 15d-J2.*

In fact, the Office leaves out key steps that are part of the logic of the rejection, which is summarized as follows: Monneret teaches that 15d-$PGD_2$ is an analog of $PGD_2$ which in turn is a precursor to 15d-J2 [which is a PPAR$\gamma$ agonist] and one agonist of another PPAR receptor [PPAR$\alpha$], clofibrate, might affect hair growth (perhaps depending on its concentration).

We have spelled out in detail why the above chain of inferences is not supported in any way by the cited references. After setting out this weak chain, the Office winds up with the following statement (Final Rejection page 3, lines 8-10), which is key to the rejection and is too unclear to warrant comment:

> *Thus the skilled artisan would have expected that 15d-PGD2, the analog of its [15d-J2's] precursor would stimulate 15d-J2 and trigger the alteration of human hair growth.*

Applicant does not understand what the Office meant to say in concluding that 15d-$PGD_2$ would "stimulate" 15d-J2. Is the Office referring to some unarticulated metabolic processes in which 15d-J2 is produced *in vivo* from 15d $PGD_2$? Is the Office simply saying that both molecules are part of metabolic cycles which include a large number of other compounds? The import of that key sentence is unclear. Without that sentence the Office articulates no basis for the rejection.

In the end, the art says absolutely nothing at all about whether 15d-$PDG_2$ inhibits hair growth.

## IV. Law and Analysis

For claims of a patent to be valid, they must define subject matter that non-obvious (35 U.S.C. § 103) in light of the "prior art," as defined by 35 U.S.C. § 102.

Applicant concedes that the two cited references were publications more than one year before its effective filing date and thus constitute prior art under 35 USC §102(b). The Office has not rejected any claims for lack of novelty.

The sole rejection here is based on obviousness, which is governed by 35 U.S.C. §103(a):

> A patent may not be obtained . . . if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains.

### A. The Elements of a Prima Facie case

More than 30 years ago the Supreme Court provided the frame work for analyzing obviousness under § 103. Obviousness must be determined by considering: (1) the scope and content of the prior art, (2) the differences between the prior art and the patent claim, (3) the level of ordinary skill in the pertinent art; and (4) (if there is a prima facie case of obviousness) secondary factors. Graham v. John Deere Co., 383 U.S. 1 (1966).

**1. Scope and Content of the Prior art**

As to the first of the factual inquiries under the Graham obviousness analysis, the scope and content of the prior art may be established by, among other things, the disclosures of references available at the time of the invention of the claimed subject matter. Relevant references include those in the same field as the claimed invention, as well as those that solve the same problem. In re Nilssen, 851 F.2d 1401, 1403 (Fed. Cir. 1988).

The scope and content of the prior art are reviewed in detail above under section "The Prior Art".

**2. Differences between the prior art and the claims**

In assessing the second factor in the obviousness analysis, the differences between the prior art and the claims at issue, it is first necessary to determine the meaning, and thus the scope, of the claims, as set forth above. See Kalman v. Kimberly-Clark Corp., 713 F.2d 760, 772 (Fed. Cir. 1983), cert. denied, 465 U.S. 1026 (1984).

In this case there does not seem to be any dispute about the meaning of the claim terms. An agonist is a molecule which operates at a receptor in a manner similar to the receptor's natural ligand, and one agonist of the DP receptor is known as 15-deoxy-$\Delta^{12,14}$ $PGD_2$ or 15d-$PDG_2$.

Applicants agree that 15d-$PGD_2$ is a DP receptor agonist, along with the other compounds listed in Tables 1 and 1A of the specification.

The above review of the Office's analysis makes it clear that any findings about hair growth in the cited art are equivocal and are limited to one particular PPAR agonist. There is no basis in the cited art to conclude that a DP agonist controls hair growth. One key difference between the art on clofibrate and the claims is that the art makes no finding that any DP agonist reduces hair growth.

The Final Rejection includes speculation not present in the art in an effort to narrow the difference between the art and the claims, but that speculation is inadequate to support the rejection.

### 3. Level of ordinary skill in the art

The level of ordinary skill in the art, the third factual inquiry, is determined by evaluating the "type of problems encountered in art; prior art solutions to those problems; rapidity with which innovations are made; sophistication of the technology; and educational level of active workers in the field." Custom Accessories v. Jeffrey-Allan Ind., 807 F.2d 955, 962 (Fed. Cir. 1986).

The level of skill in the art is reasonably high in this case.

### 4. Secondary Factors

The Office has not established a prima facie case of obviousness and the rejection must be withdrawn even without evidence of secondary factors.

## B. The need for sound analysis

In applying the Graham factors, the U.S. Patent and Trademark Office continues to have the burden of establishing a prima facie case that an applicant's claims are obvious. See In re Rouffet, 149 F.3d 1350, 1355 (Fed. Cir. 1998.

> To reject claims in an application under section 103, an examiner must show an unrebutted prima facie case of obviousness. See *In re Deuel*, 51 F.3d 1552, 1557, 34 U.S.P.Q.2d 1210, 1214 (Fed.Cir.1995). In the absence of a proper prima facie case of obviousness, an applicant who complies with the other statutory requirements is entitled to a patent. See *In re Oetiker*, 977 F.2d 1443, 1445, 24 U.S.P.Q.2d 1443, 1444 (Fed. Cir. 1992).

There is great interest in the Supreme Court's opinion in KSR Intern. Co. v. Teleflex Inc., 127 S. Ct. 1727 (2007), but KSR has not suddenly made all inventions obvious and it has not removed the requirement that Office's provide a sound analytical basis for the obviousness rejection. An important aspect of the obviousness analysis has always been the need for a rationale to support the rejection. E.g., MPEP 2144.02 ("when an Office relies on a scientific theory, evidentiary support for the existence and meaning of that theory must be provided"); and MPEP 2144.03 ("Official notice unsupported by documentary evidence should only be taken by the Office where the facts asserted to be well-known, or to be common knowledge in the art are capable of instant and unquestionable demonstration as being well-known" and "Ordinarily,

there must be some form of evidence in the record to support an assertion of common knowledge.").

In Takeda Chemical Industries Ltd. v. Alphapharm Pty., 492 F.3d 1350 (Fed. Cir. 2007), a drug for treatment of diabetes was alleged to be obvious on the grounds that the prior art divulged numerous similar compounds, making the compound used in the drug "obvious to try." The Federal Circuit found that the drug was non-obvious, as the prior art 'taught away' from the compound used by Takeda and that "obvious to try" is limited to situations where there are a finite number of identified, predictable solutions.

In In re Sang Su Lee, 277 F.3d 1338 (Fed. Cir. 2002), the Federal Circuit held that "[c]ommon knowledge and common sense," even if assumed to derive from the agency's expertise, do not substitute for authority when the law requires authority. Similarly, in In re Zurko, 258 F.3d 1379, 1386 (Fed. Cir. 2001), the Federal Court rejected the Board's arguments of common sense and basic knowledge because they were not based on evidence in the record.

> T]he Board must point to some concrete evidence in the record in support of these [obviousness] findings. To hold otherwise would render the process of appellate review for substantial evidence on the record a meaningless exercise. [Footnote omitted.]

In rejecting Applicants' arguments, the Office alleges that a mere hypothesis is a sufficient motivation or suggestion to make use of a PPAR-γ ligand to regulate hair growth and that the "claimed subject matter is the use of this suggestion" (Final Office Action, pages 3-4). Further, the Office asserts that there is a reasonable expectation of success based on Billoni's suggestion, but provides no other reasoning (Final Office Action, page 4).

Given the Office's tortured obviousness analysis detailed above, the cited art does not even support the conclusion that it would have been obvious to try 15d-$PGD_2$ to reduce hair growth.

In any event, under these circumstances, the inventive subject matter is not obvious even if it were "obvious to try". The Federal Circuit has cautioned that the "obvious-to-try" standard will be difficult to apply where the art is unpredictable See Eisai Co. Ltd. v. Dr. Reddy's Laboratory, Ltd., 533 F.3d 1533, 1559 (Fed. Cir. 2008):

> ...the Supreme Court's analysis in *KSR* presumes that the record before the time of invention would supply some reasons for narrowing the prior art universe to a "finite number of identified, predictable solutions..."
>
> In *Ortho-McNeil Pharmaceutical, Inc. v. Mylan Laboratories, Inc.*, 520 F.3d 1358, 1364 (Fed. Cir. 2008), this court further explained that this "easily traversed, small and finite number of alternatives ... might support an inference of obviousness." **To the extent an art is unpredictable, as the chemical arts often are, case law indicates that *KSR*'s focus on these "identified, predictable solutions" may present a difficult hurdle because potential solutions are less likely to be genuinely predictable**. (citations removed).

Hence, particularly in the chemical arts, when the prior art lacks "definiteness or certainty about the result", the inventive subject matter is not even "obvious to try", but in any event it is not obvious.

In this case the Office has constructed a chain of inferences that begins with an unsound conclusion about one molecule (clofibrate) and then moves on to make unsupported conclusions about other molecules and even other classes of molecules. The Office implies, but does not spell out, all the links in the chain. When those implied links are made explicit it becomes even more clear that the theory of the rejection has no basis in the art and is inadequate as a matter of law to support a prima facie finding of obviousness.

Finally, the very root of the Office's analysis is flawed because it ignores evidence from the cited art itself that supports patentability – i.e., the conclusion that clofibrate may support hair growth and the authors' reluctance to explain that contradiction. See, In re Dow Chemical Co., 837 F,2d 469, 473 (Fed. Cir. 1988), where the court held that a claim was not obvious because none of the references cited by the Office suggested that two synthetic processes could be combined successfully to produce a product with the desired properties. Indeed, at least one expert was skeptical regarding the combination before the inventors "proved him wrong". *Id.* As the court cautioned, "[e]vidence that supports, rather than negates, patentability must be fairly considered". *Id.*

For all of these reasons, the obviousness rejection must be reversed.

The brief fee of $540 is enclosed. Please apply any other charges or credits to Deposit Account No. 06-1050.

Respectfully submitted,

Date: May 7, 2009

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## Appendix of Claims

1. A method of reducing mammalian hair growth which comprises

selecting an area of skin from which reduced hair growth is desired; and

applying to said area of skin a dermatologically acceptable composition comprising an agonist of prostaglandin DP-receptor in an amount effective to reduce hair growth.

2. The method of claim 1, wherein said agonist is a prostaglandin $D_2$ analog.

4. The method of claim 1, wherein said agonist interacts strongly with the prostaglandin DP-receptor.

8. The method of claim 1, wherein said agonist is 15-deoxy-$\Delta^{12,14}$-$PGD_2$.

29. The method of claim 1, wherein the concentration of said agonist in said composition is between 0.1% and 30%.

30. The method of claim 1, wherein the composition provides a reduction in hair growth of at least 30% when tested in the Human Hair Follicle assay.

31. The method of claim 1, wherein the composition provides a reduction in hair growth of at least 60% when tested in the Human Hair Follicle assay.

32. The method of claim 1, wherein the agonist is applied to the skin in an amount of from 10 to 3000 micrograms of said agonist per square centimeter of skin.

33. The method of claim 1, wherein said mammal is a human.

34. The method of claim 33, wherein said area of skin is on the face of a human.

35. The method of claim 33, wherein the composition is applied to the area of skin in conjunction with shaving.

36. The method of claim 33, wherein said area of skin is on a leg of the human.

37. The method of claim 33, wherein said area of skin is on an arm of the human.

38. The method of claim 33, wherein said area of skin is in an armpit of the human.

39. The method of claim 33, wherein said area of skin is on the torso of the human.

40. The method of claim 1, wherein the composition is applied to an area of skin of a woman with hirsutism.

41. The method of claim 1, wherein said hair growth comprises androgen stimulated hair growth.

42. The method of claim 1, wherein the composition further includes a second component that also causes a reduction in hair growth.

43. A method of reducing mammalian hair growth, which comprises

selecting an area of skin including hair follicles from which reduced hair growth is desired; and

applying to the skin a compound selected from the group consisting of prostaglandin $D_2$, analogs of prostaglandin $D_2$, $PGJ_2$, or an analog of $PGJ_2$, in an amount effective to reduce hair growth.

44. A method of reducing mammalian hair growth, which comprises

selecting an area of skin including hair follicles from which reduced hair growth is desired; and

applying to the skin a compound that activates DP receptor signal transduction pathway in an amount effective to reduce hair growth.

45. A method of reducing mammalian hair growth, which comprises

selecting an area of skin including hair follicles from which reduced hair growth is desired; and

applying to the skin a compound that inactivates prostaglandin $D_2$ metabolic pathway in an amount effective to reduce hair growth.

## Evidence Appendix

Applicants refer the Board to the experiments of record, including the Human hair follicle growth assay at pages 11 et seq. of the specification as filed. Table III is reproduced below:

**Table III. Reduction of human hair follicle growth by $PGD_2$ and its analogs.**

| | | Hair follicle length increase (mm) | | |
|---|---|---|---|---|
| Prostaglandin | Dose ($\mu$M) | Treated | Control | % Reduction |
| $PGD_2$ | 30 | 0.24 ± 0.10 | 1.89 ± 0.38 | 87.3 ± 5.0 |
| 15-Deoxy-$\Delta^{12,14}$-$PGD_2$ | 50 | 0.10 ± 0.08 | 1.76 ± 0.27 | 94.3 ± 4.5 |
| 16,16-Dimethyl $PGD_2$ | 50 | 0.10 ± 0.05 | 1.76 ± 0.27 | 94.3 ± 2.8 |
| 15(S)-15-Methyl $PGD_2$ | 100 | 0.08 ± 0.06 | 1.10 ± 0.35 | 92.7 ± 5.5 |
| 17-Phenyl trinor $PGD_2$ | 50 | 0.10 ± 0.12 | 1.62 ± 0.43 | 93.8 ± 7.4 |
| 11-Deoxy-11-methylene $PGD_2$ | 50 | 1.11 ± 0.14 | 1.76 ± 0.27 | 36.9 ± 8.0 |
| 15(R)-15-Methyl $PGD_2$ | 50 | 0.04 ± 0.05 | 1.62 ± 0.43 | 97.5 ± 3.1 |

## Related Proceedings Appendix

None